RHEUMATOLOGY NURSE PRACTICE

Inside this Issue

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- What effects do common medications used to treat rheumatoid arthritis (RA) have on the cardiovascular system?
- Why are the use of glucocorticoids and NSAIDs dangerous in the treatment of RA patients at risk for developing cardiovascular disease (CVD)?
- What models are available to improve multidisciplinary care and reduce the risk of the development of CVD in patients with RA?
- What is self-efficacy, and why it is important to assess in patients with RA?

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Learning Objectives

1. *List the leading traditional and disease-specific risk factors that contribute to the development of cardiovascular disease (CVD) among patients with rheumatoid arthritis (RA)*
2. *Identify key questions necessary to ask every RA patient about their individual risk of developing CVD*
3. *Discuss the potential clinical utility of new CV risk assessment tools specifically designed for patients with RA*
4. *Determine the most appropriate medical interventions for RA patients who have moderate or high risk of developing CVD*

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Managing Cardiovascular Disease in Patients with Rheumatoid Arthritis

Cardiovascular disease (CVD) is the leading cause of death among patients with rheumatoid arthritis (RA). Compared with adults without RA, patients with RA are twice as likely to develop CVD and face an increased risk of developing acute coronary syndrome (ACS), heart failure, and other CVD events.[1] The high burden of cardiovascular morbidity and mortality in RA patients is attributable to a complex mix of traditional CVD risk factors and RA-specific disease characteristics.[2,3] As an independent predictor of CVD, RA is equivalent to type 2 diabetes mellitus in elevating the lifetime risk of CVD.[4]

Although CVD risk factor management has historically fallen outside the scope of care within rheumatology practices, providers are increasingly tasked with CVD risk factor screening and management. Recent guideline updates from the American College of Rheumatology (ACR) and European League Against Rheumatism (EULAR) provide a framework to assist rheumatology providers in managing CVD risk in patients with RA.[5-7]

Cardiovascular Disease Burden in RA

Patients with RA develop CVD at approximately twice the rate of individuals in the general population.[1] The elevated risk of CVD is particularly underappreciated in women with RA, who do not fit the traditional CVD risk profile.[8] Among more than 160,000 postmenopausal women participating in the Women's Health Initiative, women with RA (N=9,988) were 1.5-times more likely to develop coronary heart disease (CHD) and 2.5-times more likely to die from CVD than women without RA.[9] Compared to adults without RA, patients with RA face a 40% increase in the rate of non-ischemic heart failure and an 80% increase in the rate of ischemic heart failure.[10]

After having an initial CVD event such as a heart attack, patients with RA face a more difficult recovery and worse prognosis. A recent population-based study examined cardiovascular outcomes following an ACS event in patients with RA (n=1135) and without RA (n=3184).[11] Despite similar rates of treatment with standard cardiovascular medications in the two groups, patients with

ACTIVITY SUMMARY

In this issue of Rheumatology Nurse Practice, we explore the unique risk factors that contribute to the development of CVD in patients with RA. We also highlight a new tool for assessing RA-related CVD risk and summarize the current recommendations and best practices for CVD risk management in patients with RA.

RA had a 30% increase in the risk of recurrent ACS events during the first year of follow-up. Furthermore, compared with ACS patients without RA, ACS patients with RA had a 60% increase in the risk of 1-year mortality following their first cardiovascular event.

Improving CVD outcomes for patients with RA will require a deeper understanding of the underlying causes of RA-related CVD risk, better screening tools to identify at-risk patients, and improved management of traditional and RA-specific risk factors.

Contributing Factors to Cardiovascular Disease in RA

Three major contributing factors influence the risk of CVD in patients with RA: traditional CVD risk factors, systemic inflammation, and RA medications (Figure 1).[12] While traditional risk factors and inflammation generally increase the risk of CVD, the relationship between RA medications and CVD risk is more complex. Some medications reduce CVD risk by controlling inflammation and modifying traditional risk factors, while others exacerbate CVD risk. Each of these contributing factors is reviewed in detail in the following sections.

Traditional Cardiovascular Risk Factors

Standard CVD risk factors are highly prevalent and poorly controlled in patients with RA. In a study of 644 RA patients without diagnosed CVD, systematic risk factor screening revealed high rates of multiple CVD risk factors: smoking (24.5%), hypertension (35.8%), elevated total cholesterol (65.6%), elevated low-density lipoprotein cholesterol (LDL-C; 55.4%), low high-density lipoprotein cholesterol (HDL-C; 12.1%), elevated body mass index (BMI; 63.8%), and low physical activity rate (64.9%). In total, 20% of these RA patients had a high 10-year risk of a fatal CVD event (≥5%).[13] Despite an increased risk of CVD events, patients with RA are rarely targeted for CVD risk assessment in the primary care or rheumatology settings.[14]

Role of Systemic Inflammation

The systemic inflammation that characterizes RA disease activity adversely affects many organ systems in the body, including the cardiovascular system. In patients with RA, the cumulative burden of systemic inflammation directly correlates with CVD risk. Patients with more severe RA disease activity have a higher risk of CVD, as do patients with a longer duration of RA.[3] Many of the markers of inflammation in RA, including C-reactive protein (CRP), interleukin (IL)-6, and tumor necrosis factor (TNF), also independently predict an increased risk of CVD.[15,16] Increased serum TNF-α levels correlate with a 3-fold increase in the risk of recurrent myocardial infarction (MI) or coronary death.[17]

Atherosclerosis is an inflammatory form of CVD characterized by endothelial dysfunction, plaque rupture, and thrombosis.[18] Even in the absence of traditional CVD risk factors, the systemic

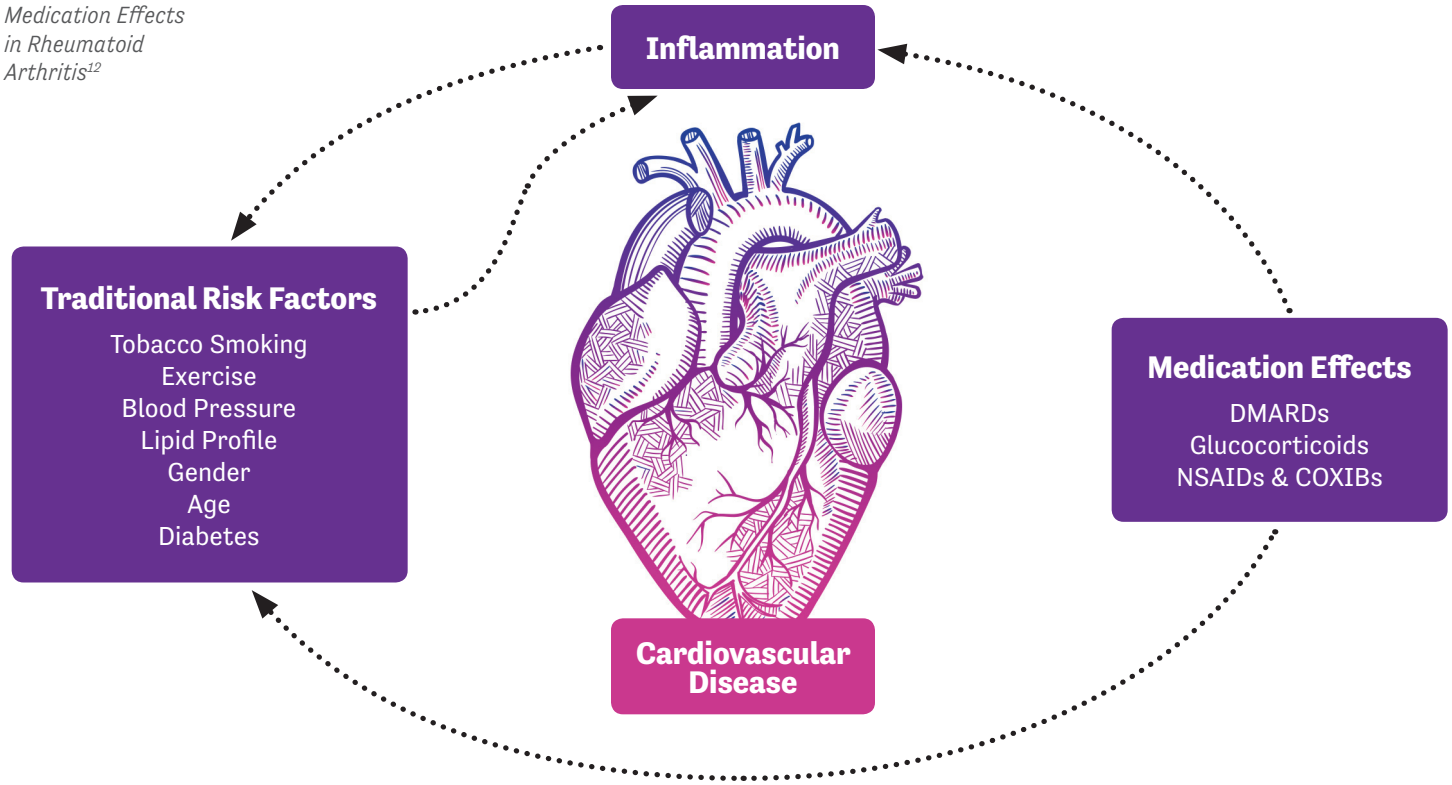


Figure 1
Traditional Risk Factors, Systemic Inflammation, and Medication Effects in Rheumatoid Arthritis[12]

inflammation of RA is sufficient to drive the development of atherosclerosis. In one recent study of patients without traditional CVD risk factors, carotid ultrasound detected subclinical atherosclerosis in 27.5% of RA patients and 10% of healthy controls (P=0.04). In the RA group, atherosclerosis was more commonly detected in those with higher RA disease activity (P=0.02), erosive disease (P=0.06), and RF-positive disease (P=0.03).[19]

Medication Effects: Glucocorticoids

High-dose and/or long-term glucocorticoid use can adversely affect CVD risk factors such as lipids, blood pressure, obesity, glucose tolerance, and insulin resistance.[7] In a large database analysis of RA patients (N=34,619), the use of prednisolone 4 mg/day—or an equivalent glucocorticoid dose—within the last 6 months was associated with a 48% increase in the risk of diabetes.[20]

Historically, glucocorticoids have been widely used in patients with RA. However, with the availability of effective non-biologic and biologic disease-modifying antirheumatic drugs (DMARDs), patients and providers have safer options for symptom management. Given the risk of exacerbating CVD risk factors with glucocorticoids, there has been a shift in RA management away from relying on the analgesic benefits of these agents.[7] According to the 2015 ACR guidelines, glucocorticoids should be used only as a bridge to provide short-term anti-inflammatory effects in patients who are starting DMARD therapy, or in patients with RA flares.[5] Furthermore, glucocorticoid therapy should be restricted to the lowest possible dose (≤10 mg/day of prednisone or equivalent) for the shortest possible duration (<3 months) to maintain a favorable risk/benefit ratio.[5]

Despite recommendations to minimize glucocorticoid use, these agents remain widely used in patients with RA, particularly in the primary care setting. In another large database study of RA patients (N=16,536), 47% were prescribed oral glucocorticoid therapy by their primary care provider (PCP).[21] Moreover, glucocorticoids were often used in doses that exceeded the recommended threshold of low-dose therapy. Among RA patients who were prescribed glucocorticoids, more than 50% were prescribed >10 mg/day and 20% were prescribed >30 mg/day.[21] Therefore, it is important to ask RA patients if they are using any glucocorticoids, including any prescribed by their PCP (see **10 Questions to Ask Your RA Patient About CVD**, p. 6).

Medication Effects: NSAIDs

The nonsteroidal antiinflammatory drugs (NSAIDs) are a diverse family of agents used to control pain and inflammation in patients with RA. In general, individual NSAIDs are classified according to their mechanism of action and whether they selectively target the cyclooxygenase (COX)-1 or COX-2 isoforms. Nonselective NSAIDs act on both COX-1 and COX-2.

Treatment with nonselective NSAIDs and selective COX-2 inhibitors increases the risk of CVD events, although the degree of increased risk varies considerably from agent to agent.[7,22] One meta-analysis evaluated the cardiovascular safety of 7 NSAIDs (celecoxib, diclofenac, etoricoxib, ibuprofen, lumiracoxib, naproxen, and rofecoxib) in patients with RA, osteoarthritis, and other indications for NSAID therapy (N=116,429).[22] As a class, NSAID use increased the risk of CVD events 2-4 times higher than placebo. Individual NSAIDs showed particularly strong adverse associations with specific CVD outcomes, including MI (rofecoxib and lumiracoxib), stroke (ibuprofen and diclofenac), and death (etoricoxib and diclofenac). Among all NSAIDs analyzed, naproxen was the least harmful in terms of cardiovascular safety.[22] In an observational study of real-world RA patients (N=17,320), diclofenac and rofecoxib significantly increased the risk of CVD, but other NSAIDs did not appear to exacerbate RA-related CVD risk.[23]

The interaction between NSAIDs and CVD risk is concerning in patients with RA, who already carry an increased baseline risk of CVD.[7] EULAR guidelines recommend caution in using NSAIDs and COX-2 inhibitors in patients with RA, particularly those with other CVD risk factors or established CVD.[7]

Evaluating Cardiovascular Risk in the Rheumatology Setting

Managing cardiovascular risk in patients with RA begins with routine screening and documentation of cardiovascular risk factors. Standard CVD risk-assessment tools such as the Framingham Risk Score (FRS) and Systematic Coronary Risk Evaluation (SCORE) algorithm underestimate the risk of CV events in patients with RA.[3,24] Researchers at Brigham and Women's Hospital in Boston recently undertook the task of developing a CV risk score specific to patients with RA. The result—the Extended Risk Score-Rheumatoid Arthritis (ERS-RA)—is the first risk score that accounts for the unique CV risk profile of patients with RA.[3]

Developing the ERS-RA

The first step in developing the ERS-RA was to better understand which disease-specific risk factors increased CV risk in patients with RA, and by how much. Researchers turned to the

Consortium of Rheumatology Researchers of North America (CORRONA) database, a U.S.-based registry that includes more than 40,000 patients with RA. Using CORRONA data from 15,744 patients with detailed CV risk information, researchers examined multiple potential predictors of CV risk, including the following:

- RA disease activity, measured by the Clinical Disease Activity Index (CDAI)
- Extent of disability, measured by the Health Assessment Questionnaire Disability Index (HAQ-DI)
- Duration of RA
- Serology, including rheumatoid factor (RF) and anti-cyclic citrullinated peptide antibody (ACPA)
- Presence of joint erosions and subcutaneous nodules
- Use of medications, including oral corticosteroids, methotrexate (MTX), tumor necrosis factor (TNF) inhibitors, and nonsteroidal anti-inflammatory drugs (NSAIDs)

Of the RA disease features tested, 4 risk factors significantly worsened CV risk in patients with RA. **Daily prednisone use**, compared with non-use, increased the likelihood of CV events by 61% (HR, 1.61). A **disease duration** of longer than 10 years increased CV risk by 43% (HR, 1.43). Patients whose CDAI scores indicated **moderate or high disease activity** had a 31% increase in the risk of CV events compared to patients with low disease activity or those in clinical remission (HR, 1.31). Finally, **moderate or high disability** according to the HAQ-DI score was associated with an 18% increase in CV risk compared with low or no disability (HR, 1.18).

Based on these findings, the ERS-RA risk calculator prompts clinicians to indicate—yes or no—whether the following risk factors are present: current prednisone use, RA disease duration ≥10 years, CDAI score >10, and HAQ-DI score >0.5. The tool combines the 4 RA-specific items with 6 traditional risk factors for CV events, including age, gender, hypertension, diabetes, dyslipidemia, and smoking. The ERS-RA then uses an algorithm that assigns different weight to different risk factors to calculate a patient's 10-year risk of experiencing a CV event.

The ERS-RA is available online for free download and use at the Brigham and Women's website (www.brighamandwomens.org) by entering "ERS-RA risk calculator" in the Search field.

Improved CV Risk Prediction

Once the ERS-RA calculator was developed, the next step involved validating the tool in a real-world patient population.[3] Again researchers turned to the CORRONA database, where they assessed baseline CV risk in another cohort of RA patients (n=7,861) using both the ERS-RA and a traditional FRS model. Patients were classified into 1 of 4 risk categories based on their estimated 10-year risk of cardiovascular events:

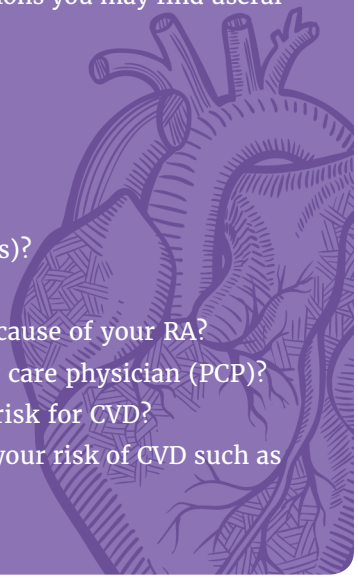
- <5% risk
- 5% to <10% risk
- 10% to <20% risk
- ≥20% risk

After observing patients for an average of 2.9 years, researchers were able to evaluate the accuracy of the baseline CV risk scores in predicting CV events. The expanded ERS-RA risk score correctly described the risk of CV events in 17% more patients than the traditional FRS calculator

10 Questions to Ask Your RA Patient About CVD

The ERS-RA risk calculator estimates the 10-year risk of CVD in RA patients based on 10 risk factors.[3] These include both traditional cardiovascular risk factors as well as RA-specific considerations. Although the ERS-RA is still under development for clinical use, the tool provides a useful guide for starting a conversation with RA patients about their CVD risk. Here is a brief list of questions you may find useful in starting a discussion.

1. Do you smoke?
2. Do you have diabetes?
3. Do you have high cholesterol?
4. Do you have high blood pressure?
5. Are you using any glucocorticoids (steroids)?
6. How long have you had RA?
7. Do you have any functional limitations because of your RA?
8. When was last time you saw your primary care physician (PCP)?
9. Has your PCP talked with you about your risk for CVD?
10. Are you taking any medications to reduce your risk of CVD such as low-dose aspirin or a statin?



CVD Risk Prediction in Sample RA Patients

Cardiovascular risk scores can inform decisions about the need for lifestyle modifications and other interventions to reduce cardiovascular risk. For instance, the American College of Cardiology/American Heart Association (ACC/AHA) recommends starting lipid-lowering statin therapy in patients whose 10-year predicted risk of CVD events is 7.5% or higher.[25] To illustrate how risk scores may influence clinical decision-making in RA patients, the ERS-RA research team described 2 sample case scenarios (Figure 2).[3]

Case 1. The first patient is a 55-year-old woman with the following medical history:

- RA disease duration >10 years
- Current use of corticosteroids
- Moderate RA disease activity (CDAI >10)
- Moderate RA-related disability (MHAQ-DI >0.5)
- History of hypertension
- No history of diabetes, hyperlipidemia, or tobacco use

Using the ERS-RA risk calculator, her 10-year risk of experiencing a CV event is 9.3%. This exceeds the ACC/AHA 10-year risk threshold of 7.5%, indicating that she is a candidate for statin therapy. However, using a traditional CV risk calculator that does not account for corticosteroid use or RA disease activity, her estimated 10-year risk is 4.5%, below the recommended threshold for statin use.[3]

Case 2. The second case example involves a 50-year old male patient with the following disease characteristics:

- RA disease duration ≤10 years
- No history of corticosteroid use
- Low RA disease activity (CDAI ≤10)
- Minimal RA-related disability (MHAQ-DI ≤0.5)
- History of hypertension
- No history of diabetes, hyperlipidemia, or tobacco use

According to the ERS-RA risk calculator, the patient's 10-year risk of a CV event is 4.3%. Therefore, his risk falls below the ACC/AHA recommended threshold for lipid-lowering therapy. This case illustrates that a patient may have both active RA and a traditional CVD risk factor such as hypertension, yet have disease that is mild enough not to warrant additional risk-reducing medications. Regardless of ERS-RA risk score, all patients with RA should be managed with the goal of controlling RA disease activity and minimizing CV risk (see later section on Co-Management Principles).[3]

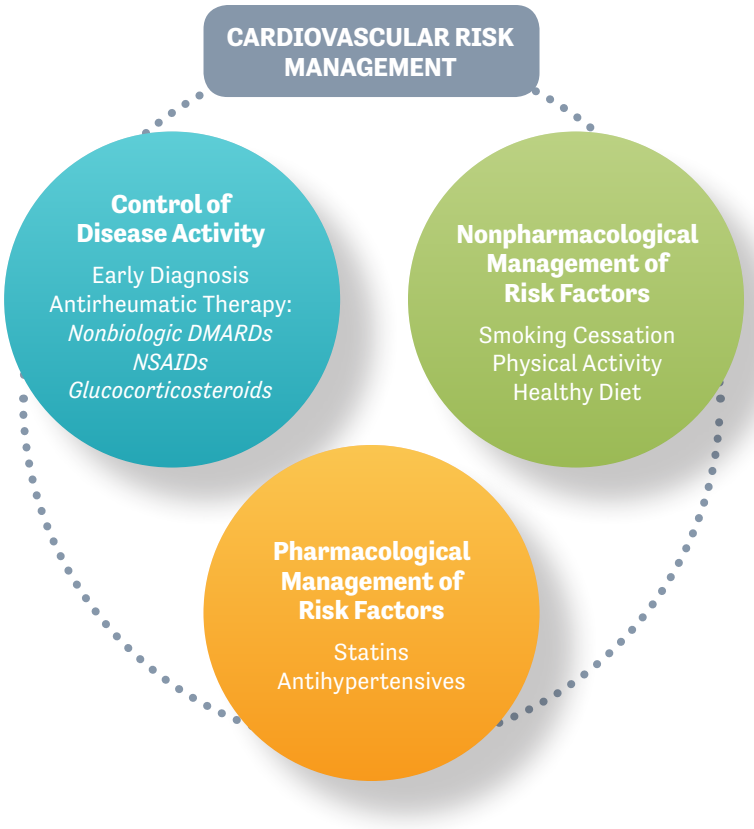
Figure 2
ERS-RA Risk Score Calculator in Sample Patients with Rheumatoid Arthritis

Risk Factor	Acceptable Range	RA Patient A	RA Patient B
Age	20-80	55	50
Gender	M=Male, F=Female	F	M
Diabetes	Y=Yes; N=No	N	N
Hyperlipidemia	Y=Yes; N=No	N	N
Hypertension	Y=Yes; N=No	Y	Y
Current tobacco use	Y=Yes; N=No	N	N
CDAI	Y if CDAI>10; N if CDAI≤10	Y	N
mHAQ-DI > 0.5	Y if mHAQ-Di>0.50; N if mHAQ-DI≤0.5	Y	N
Prednisone use	Y=Current Use; N=No Current Use	Y	N
RA Disease duration ≥ 10 y	Y if >10 yrs; N if ≤10 yrs	Y	N
10-Year ERS-RA Risk Score		**9.3%**	**4.3%**

CVD Risk Management Principles

Managing CV risk in patients with RA involves a 3-pronged strategy: 1) controlling the systemic inflammation of RA; 2) lifestyle modifications; and 3) cardiovascular medications (Figure 3).[12]

Figure 3
Managing Cardiovascular Risk in Patients with RA[12]



Controlling RA-Related Systemic Inflammation

The primary goal of treatment in patients with RA is to control the chronic inflammatory disease activity that drives joint symptoms and extraarticular manifestations. As illustrated in the ERS-RA risk score algorithm (Figure 2), providing better control of RA disease activity can reduce CVD risk. In addition, some RA medications demonstrate direct beneficial effects on CVD risk factors and cardiovascular outcomes in patients with RA (Table 1).

Methotrexate

Methotrexate (MTX) is often used to control the chronic systemic inflammation of RA, with additional favorable effects on CVD outcomes.[26] In a study of 317 patients with RA, treatment with high-dose MTX (≥20 mg/wk) was associated with reduced markers of atherosclerosis, including reduced intima-media thickness and fewer atherosclerotic plaques in the carotid and femoral arteries.[27] In another study of patients with RA or psoriasis, MTX treatment for at least 1 year was associated with a 29% reduction in ischemic CVD events, including angina, acute coronary syndrome, coronary revascularization, stroke, and CVD death.[28] In a long-term study of patients with RA, treatment with MTX reduced the risk of CVD deaths by 70% compared with no MTX use over the 6-year follow-up period.[29]

Folic acid supplementation is recommended during treatment with MTX to correct elevated plasma homocysteine levels, one of the main side effects of MTX treatment.[4,30] MTX is a folic acid antagonist that disrupts several metabolic pathways and results in folic acid depletion.[30] Decreased levels of folic acid can cause plasma homocysteine to accumulate, resulting in hyperhomocysteinemia. In addition to being a marker of systemic inflammation, hyperhomocysteinemia independently predicts an increased risk of CVD.[30,31] Folic acid supplementation keeps homocysteine levels in balance during MTX treatment and may contribute to CVD prevention in patients with RA.[32]

Other Nonbiologic DMARDs

In addition to MTX, several other nonbiologic DMARDs exert beneficial effects on CVD risk in patients with RA.[33] In a study of 4,363 patients with RA, leflunomide reduced CVD morbidity by 41% and sulfasalazine by 8%.[34] Hydroxychloroquine is associated with favorable lipid effects in patients with RA, including reduced LDL-C levels, reduced triglyceride levels, improved LDL-C/HDL-C and total cholesterol/HDL-C ratios, and an increased likelihood of reaching lipid targets.[35,36]

Anti-TNF Therapies

Multiple studies have confirmed the benefits of anti-TNF therapy on CVD endpoints in patients with RA. In one study of patients with RA who started treatment with either MTX + anti-TNF therapy or MTX + a nonbiologic DMARD, patients in the anti-TNF group had a 29% reduction in the risk of MI, stroke, or coronary revascularization after 6 months compared with those who added a nonbiologic DMARD.[37] In another study of RA patients treated with or without TNF-targeted therapy, anti-TNF therapy was associated with a 55% reduction in the risk of coronary artery disease.[38]

The duration of anti-TNF therapy appears to be linked to the magnitude of cardioprotective benefits in patients with RA. In a large claims

Drug Class	Effects on CVD Risk	ACR/EULAR Recommendations[5,7]
Glucocorticoids	Adverse effects on lipids, BP, obesity, glucose tolerance, insulin resistance[7]	If needed, give at the lowest dose (≤10 mg/day of prednisone or equivalent) for the shortest duration (<3 months) possible
NSAIDs	Increased risk of CV events, including MI, stroke, CV death[22,23]	Use nonselective NSAIDs and COX-2 inhibitors with caution
Methotrexate	Reduced risk of CVD and CV death[29,69]	Recommended to control inflammatory disease activity and to lower CV risk; give with folic acid supplements to avoid hyperhomocysteinemia
Other nonbiologic DMARDs (e.g., hydroxychloroquine)	Reduced risk of cardiovascular morbidity and improved lipid profiles[34–36]	No specific recommendations
Anti-TNF therapies	Reduced CV risk cardiovascular morbidity and mortality[38,70]	Recommended to reduce systemic inflammation and to lower CV risk; avoid use in patients with NYHA Class III/IV heart failure
B-cell-targeted therapy (e.g., rituximab)	Reduced LCL-C levels, reduced risk of atherosclerosis, improved endothelial function, reduced markers of arterial stiffness[33,44,45]	No specific recommendations; preferred over anti-TNF therapy in patients with NYHA Class III/IV heart failure
T-cell targeted therapy (e.g., abatacept)	Increased HDL-C levels, reduced risk of myocardial infarction, and improved insulin sensitivity[41–43]	No specific recommendations; preferred over anti-TNF therapy in patients with NYHA Class III/IV heart failure
Anti-IL-6 therapy (e.g., tocilizumab)	Reduced markers of inflammation (CRP) reduced markers of arterial stiffness, and improved markers of cardiac function[33,44,46]*	No specific recommendations; preferred over anti-TNF therapy in patients with NYHA Class III/IV heart failure
Small molecule therapy (e.g., tofacitinib)	Reduced arterial stiffness when used in combination with MTX[48]*	No specific recommendations; preferred over anti-TNF therapy in patients with NYHA Class III/IV heart failure

* *Treatment with tocilizumab and tofacitinib is associated with moderately increased HDL-C and LDL-C levels, although the implications on CVD risk are unclear.*[71]

Table 1
Cardiovascular Effects of Rheumatoid Arthritis Medications

database of patients with RA (N=113,677), longer exposure to anti-TNF therapy was associated with a significantly greater reduction in CVD event risk.[39] Compared with no anti-TNF use, each 6-month increment of anti-TNF therapy reduced CV event risk by 12%. After 3 years, patients treated with anti-TNF therapy experienced a 51% reduction in CV event risk compared with patients who were treated with other RA medications, including MTX and other non-biologic DMARDs.

Although the mechanisms driving the cardioprotective effects of anti-TNF therapies are unclear, these agents appear to improve endothelial function in patients with RA.[40] According to current EULAR guidelines, using anti-TNF therapy in combination with MTX to reduce the chronic systemic inflammation of RA is an effective strategy for reducing CVD risk in patients with RA.[7]

Precaution with Use of Anti-TNFs in Patients with Heart Failure

Due to reports of worsening heart failure during treatment with anti-TNF therapy, the 2015 ACR guidelines caution against the use of anti-TNF therapy in patients with New York Heart Association (NYHA) class III or IV heart failure.[5] Class III heart failure describes patients who have no symptoms at rest, but marked limitation in physical activity as well as fatigue, palpitation, dyspnea, or angina with less-than-normal physical exertion. Class IV heart failure describes patients who have heart failure symptoms even at rest, and who cannot carry on any physical activity without discomfort.[5]

According to the ACR guidelines, the preferred treatment options for RA patients with

moderate-to-severe RA disease activity and class III/IV heart failure include:[5]

- Combination DMARD therapy (e.g., methotrexate and sulfasalazine)
- Non-TNF biologic therapy (e.g., abatacept, rituximab, or tocilizumab)
- Tofacitinib, the oral small-molecule Janus kinase (JAK) inhibitor

Other Biologic DMARDs

A growing body of evidence supports the beneficial effects of non-TNF biologic DMARDs on CVD risk factors and cardiovascular events in patients with RA. Abatacept is associated with increased HDL-C levels, improved insulin sensitivity, and reduced risk of MI relative to treatment with anti-TNF agents in patients with RA.[37-39] Rituximab, an anti-CD20 antibody that targets B cells, has been shown to reduce endothelial dysfunction, markers of arterial stiffness, and atherosclerosis.[34-36] Treatment with tocilizumab, an anti-IL-6 monoclonal antibody, is also associated with reduced markers of arterial stiffness and improved markers of cardiac function in RA patients.[33,44,46]

To date, few studies have examined the cardiovascular effects of tofacitinib, the newest non-TNF small molecule option for RA management. However, tofacitinib has shown consistent safety and efficacy in RA patients with CVD risk factors such as hypertension, dyslipidemia, obesity, and diabetes.[47] Tofacitinib also reduces arterial stiffness when used in combination with MTX.[48]

Lifestyle Modifications: Physical Activity

Physical activity is strongly associated with beneficial effects on CVD risk factors in patients with RA. In a study of 165 adults with RA, patients who reported higher levels of weekly physical activity had a significantly better heart rate, waist-to-hip ratio, systolic blood pressure, and HDL particle concentration.[49] The cardiovascular benefits of exercise were confirmed in a randomized trial in which patients with RA were assigned to participate in a supervised exercise program (n=28) or continue with usual RA care (n=24).[50] After 3 months, patients in the exercise group showed significant improvements in several CVD risk factors compared with baseline, including decreased waist circumference (2.8% reduction; $P<0.0001$), improved aerobic capacity (19% improvement; $P=0.002$), and decreased C-reactive protein levels (32% reduction; $P=0.025$). Beyond CVD risk factors, patients in the exercise group also experienced significant improvements in grip strength, fatigue scores, and cognitive function. By comparison, patients in the control group experienced no significant changes in their CVD risk factors over the 3-month study period.

The 2015 EULAR guideline on CVD risk management recommends exercise as an essential component of RA management, both to improve RA outcomes and lower CVD risk.[6] Regarding exercise targets, the AHA and the ACR recommend a goal of at least 150 minutes of moderate exercise per week, or 75 minutes of vigorous exercise per week.[51,52] To reach this goal, patients with RA may choose to perform 30 minutes of moderate exercise per day, 5 days per week. Importantly, shorter periods of just 10 to 15 minutes are beneficial for RA patients with physical limitations who need to work up to the weekly goal.[51,52]

Lifestyle Modifications: Smoking Cessation

Tobacco use is an independent risk factor for CVD in patients with RA and in the general population.[3] Smoking is also associated with worse RA-related outcomes, as current smokers have more severe RA disease activity than nonsmokers, are less responsive to treatment with MTX and anti-TNF therapy, and are less likely to achieve clinical remission.[53,54] The EULAR guidelines recommend that all patients with RA receive counseling about smoking cessation, regardless of the presence of other CVD risk factors.[7]

Cardiovascular Medications in Patients with RA: Low-Dose Aspirin

Although aspirin is classified as an NSAID, its mechanism of action varies by dose. Low-dose aspirin (50-81 mg/day) acts as a selective COX-1 inhibitor that targets platelets to exert an antithrombotic effect. By comparison, the dose of aspirin must be increased 100-fold to induce an anti-inflammatory effect. High-dose aspirin (3-5 g/day) inhibits COX-2 and targets inflammatory cells to reduce swelling and pain in patients with RA.[55]

Low-dose aspirin is commonly recommended for the prevention of CVD in certain high-risk individuals in the general population. In 2015, the U.S. Preventive Services Task Force published updated draft guidelines recommending low-dose aspirin (75-100 mg/day) for the primary prevention of CVD in all adults aged 50 to 59 whose 10-year risk of CVD is ≥10%, and for older patients with this degree of risk on an individualized basis.[56] To prevent gastrointestinal injury, patients taking daily low-dose aspirin may benefit from treatment with a proton-pump inhibitor (PPI), histamine 2-receptor antagonists (H2RA), or mucoprotective

(MP) drug. However, these protective agents are widely underutilized. In one study of aspirin prescribing patterns, only 3.5% of low-dose aspirin prescriptions were accompanied by a prescription for PPIs, H2RAs, and MPs.[57]

The potential cardiovascular benefits of low-dose aspirin in patients with RA are unclear. One recent study examined the association between aspirin use and CVD events in RA patients aged >60 years who had experienced an MI (N=705) between 1995 and 2013.[58] In the analysis, "aspirin use" was defined as any exposure to low-dose aspirin within 7 days prior to the RA patient's first MI. After controlling for the use of other medications that might alter cardiovascular risk, aspirin had no effect on the risk of MI or any other CVD events. In addition, there was no association between aspirin and MI risk when the definition of "aspirin use" was extended to 15 days or shortened to 0 days prior to the first MI, or when the analysis was restricted to male patients alone. These findings highlight the challenges of CVD risk modification in patients with RA.

The 2015 EULAR guidelines acknowledge the lack of current evidence supporting aspirin use to prevent CVD in patients with RA and conclude that "the use of aspirin for the primary prevention of cardiovascular events in patients with inflammatory joint diseases is not recommended."[6]

Cardiovascular Medications in Patients with RA: Statins

Statin therapy is a cornerstone of CVD risk reduction in all individuals with elevated lipid levels, including patients with RA. In 2015, EULAR published updated guidance on CV risk management reaffirming the central role of statin therapy for patients with RA.[6] Despite these recommendations, statin therapy is underutilized among RA patients. In a recent study that applied current ACC/AHA recommendations for statin therapy to a cohort of statin-naïve adults with RA (N=677), 38.8% of women and 78.5% of men were candidates for starting statin therapy based on their elevated 10-year risk of CVD events (≥7.5%).[59]

Historically, one of the barriers to statin use in patients with RA has involved the lack of specific guidance on optimal statin use in this patient population. However, new clinical trial results and guideline recommendations provide a new framework for lipid management in patients with RA. The TRACE-RA (Trial of Atorvastatin in the Primary Prevention of Cardiovascular Endpoints in Rheumatoid Arthritis) study examined the role of statin therapy in reducing cardiovascular events in patients with RA.[60] The trial enrolled 2,986 patients with RA who had additional risk factors for CVD, including older age (>50 years) and long duration of RA (>10 years). Patients were randomly assigned to treatment with atorvastatin 40 mg daily or placebo. All patients also received advice about lifestyle modifications for reducing CV risk. After a median follow-up of 2.5 years, patients in the statin group had a significantly greater reduction in LDL-C (mean reduction, 41.4 mg/dL) compared with patients who received lifestyle advice alone (mean reduction, 5.4 mg/dL) (P<0.001). Patients in the statin group also had a 34% reduction in the risk of a major CV event compared with the placebo group, although the difference did not reach statistical significance (P=0.119). Findings from the TRACE-RA trial support the use of statins in patients with RA, in accordance with EULAR recommendations.

In 2015, the National Lipid Association (NLA) published updated guidelines on lipid management in a range of patient populations, including RA.[61] Key lipid-management recommendations for patients with RA include the following:

- Statins are appropriate first-line treatment for dyslipidemia in patients with RA.
- Cholesterol goals for patients with RA are the same as those for the general population, provided that RA is counted as an additional risk factor when stratifying CVD risk.
- Patients with RA should be referred to a cardiologist or other lipid specialist when lipid levels remain high despite standard statin therapy (e.g., LDL-C ≥190 mg/dL and/or non-HDL-C ≥220 mg/dL) and in cases of very high triglyceride levels (≥500 mg/dL).
- Given the increased risk of CVD in RA, it is reasonable to refer RA patients to a cardiologist or other lipid management specialist when LDL-C levels are ≥160 mg/dL.
- LDL-C levels can be artificially lowered during an RA flare. Therefore, if lipid levels are measured during a flare, they should be re-measured once RA disease activity is controlled.
- Among current RA treatments, only tofacitinib and tocilizumab have specific lipid monitoring recommendations included in the prescribing information. Lipid levels should be monitored 4-8 weeks after starting these agents, and at 24-week intervals thereafter.

Other Cardiovascular Medications

Patients with RA who require medication to manage other cardiovascular risk factors such as hypertension should be treated in accordance with standard CVD guidelines, such as those from ACC/AHA.[7] Due to the anti-inflammatory properties of angiotensin-converting enzyme (ACE) inhibitors and/or angiotensin-receptor blockers (ARBs), EULAR recommends the preferential use of these agents for controlling hypertension in patients with RA.[7]

Challenges and Opportunities for Multidisciplinary RA-CVD Care

According to current practice trends, patients with RA are not being targeted for CVD risk factor screening and management. In a recent survey of rheumatologists, only 40% reported routinely checking lipids levels in their patients with RA. By comparison, more than half of respondents reported checking lipids only "sometimes" (33%), "rarely" (21%), or "never" (4%).[62] In another study of RA patients managed at an academic rheumatology clinic, 49% of patients were missing documentation of their lipid levels, and 8% had not undergone screening for diabetes. Using current ACC/AHA risk-assessment criteria, 21% of RA patients had a 10-year risk of CVD of ≥5%. Among those who were candidates for statin therapy, only 47% were being treated with recommended statin regimens.[63]

Every member of the multidisciplinary RA care team plays a critical role in ensuring that RA-related cardiovascular disease is properly assessed and managed

Barriers to RA-CVD Risk Management

Historically, the bulk of CVD risk factor assessment and management in patients with RA has occurred at the primary care level.[64] Ideally, both RA patients and their PCPs are aware of the association between RA and CVD and apply this awareness to shared decision-making. Unfortunately, survey results indicate low levels of awareness at both the patient and provider level. In a survey of 376 PCPs, only 32% correctly identified RA as an independent risk factor for CVD.[64] In another survey of patient and provider knowledge, nearly all RA patients and half of PCPs were unaware of the RA-mediated risk of CVD.[14]

With inadequate CVD risk management at the primary care level, the responsibility for risk-factor screening and management is increasingly falling to rheumatology providers. Common barriers to screening for CVD risk factors in the rheumatology setting include the burden of screening time, the complexity of RA patients, and forgetting or needing a prompt (e.g., within the electronic health record) to screen.[62] Once risk factors have been identified, whose responsibility it is to intervene? In general, rheumatology providers are comfortable managing certain risk factors that are 'shared' between RA and CVD, such as smoking, exercise, diet, and physical activity.[14] However, rheumatology providers are reluctant to act on risk factors outside of their expertise, such as increased blood pressure and lipid levels. In a recent survey, rheumatologists described the following reasons for their reluctance to manage CVD risk factors in RA patients:[14]

- Poor knowledge of current CVD guidelines
- Vague RA-specific recommendations regarding CVD risk factor management
- Concerns about overstepping boundaries by prescribing CVD medications

Instead of managing CVD risk factors in the rheumatology clinic, rheumatologists report transferring responsibility to the PCP or to the patient. Without a formal framework for communication between PCPs and rheumatology providers, however, recommendations regarding CVD risk management are often lost to follow-up.[14]

Successful Models for Multidisciplinary Care

Every member of the multidisciplinary RA care team, including rheumatologists, advanced practice clinicians, rheumatology nurses, PCPs, and patients, plays a critical role in ensuring that RA-related CVD risk is assessed and managed. In particular, rheumatology nurses can manage RA-CVD risk through patient education, counselling, and adherence support.[65] Compared with rheumatologist consultations, rheumatology nurse consultations are highly cost effective, show no decrease in efficacy in terms of clinical outcomes, and yield higher levels of patient satisfaction.[66]

Models for successful CVD risk management in patients with RA are emerging. Rheumatologists at the National Jewish Health Medical Center in Denver have established a pilot RA-CVD clinic to facilitate risk factor assessment in patients with RA.[67] As part of the program, rheumatologists at the academic rheumatology clinic can use a prescribed order set that includes referral to the RA-CVD clinic, electrocardiogram, lipid profile, and hemoglobin A1C level measurements. Since the launch of the

program, screening for hypercholesterolemia and diabetes improved by 60% among patients with RA. In addition, 38% of patients who were assessed in the RA-CVD clinic were started on statin therapy based on their cardiovascular risk score.

Role of Patient Education

Patient education around RA-related CVD risk is an essential component of successful risk management. Patients with RA tend to underestimate their risk for CVD events, and therefore may have limited motivation to reduce their risk profile. In one study of RA patients (N=111), 53% were classified as having a very high 10-year risk of CVD (≥20%), yet only 3% of these patients believed that their risk of CVD was elevated.[68]

Additional misperceptions around CVD risk management may interfere with successful treatment outcomes. A recent study of RA patients' perceptions around CVD risk revealed several opportunities for patient education:[14]

- RA patients consider their rheumatologist to be their "main doctor," and do not seek additional health information from their PCP. Some RA patients could not name their PCP.
- Patients assume that any provider can address any health topic at any visit. Without their rheumatologist or rheumatology nurse flagging a specific issue (eg, hypertension), patients assume they have no additional health concerns.
- Patients may believe that routine laboratory tests to monitor rheumatology medications also include preventive labs such as lipid levels.
- Patients with active RA may prefer to focus on controlling their RA symptoms and are less interested in preventive care.

Patient education regarding treatment adherence is also imperative, given that patients show different levels of adherence to different risk-management interventions. In a study of patients with RA who were prescribed pharmacologic and lifestyle interventions to manage CV risk, 90% reported taking all lipid-lowering and/or blood pressure-lowering medications as prescribed. However, only 68% reported following recommended dietary advice, and 62% reported adhering to recommendations regarding exercise.[68] These findings underscore the importance of ongoing patient education about following all treatment recommendations.

Despite the importance of patient education, this component of patient care takes low priority in current rheumatology practice. In one survey, only 17% of rheumatologists reported that they "always" counsel their RA patients about the increased risk of cardiovascular disease, while 50% said that they provide counseling "most of the time." The remaining one-third of rheumatologists only sometimes (25%), rarely (4%), or never (4%) provide counseling to their RA patients about CVD risk.[62]

Summary

Patients with RA face an increased risk of CVD due to chronic RA-related systemic inflammation, a high prevalence of traditional CVD risk factors, and the adverse effects of common RA medications such as NSAIDs and glucocorticoids. Unfortunately, the management of CVD risk factors in patients with RA is often poor in the primary care setting, shifting the responsibility to rheumatology providers. The ERS-RA is a new risk score calculator designed to estimate the 10-year risk of CVD in patients with RA by accounting for both traditional and RA-specific risk factors. With additional testing, the ERS-RA risk calculator may be an effective tool for identifying patients with RA who have an increased risk of CVD and require additional interventions to lower their long-term risk of CVD morbidity and mortality. Reducing systemic inflammation with the effective use of RA medications is the first step in CVD risk factor management in patients with RA. Lifestyle modifications are also important to improve RA-specific outcomes and reduce CVD risk. Some patients with RA may require treatment with statins or other cardiovascular medications for additional risk-factor modification. Multidisciplinary collaboration between rheumatologists, advanced practice clinicians, rheumatology nurses, PCPs, and patients is essential for managing RA-related CVD risk.

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The "What Ifs" That Stay With Us

by Iris Zink, MSN, NP

Sometimes, we encounter a patient in our practice who touches our life and our soul, and never leaves our heart. One such patient of mine was Michael.

Michael fit the profile of our usual stoic patient with rheumatoid arthritis (RA). He was a hard-working carpenter from a family line of carpenters. He had worked hard all his life in the family business. He was a jovial patient who rarely complained and was passionate about his family.

In 1999, at the age of 43, Michael started having hand and wrist pain that limited his ability to swing his hammer. That same year, he had his right wrist fused due to damage in that wrist. Following the surgery, he was initially sent to our practice for a full workup.

After our examination, we diagnosed Michael with seronegative RA. His disease eventually progressed to the point where we had him on a regimen of methotrexate, leflunomide, hydroxychloroquine, and adalimumab. He was also taking 10–15 mg of prednisone daily to be able to continuing working and support his family.

Unfortunately, we were never able to get Michael's disease into remission. There were several times he came in for checkups and told us that the adalimumab worked great for the first few days before all of his symptoms returned.

Michael's therapy was also frequently interrupted by infections and surgeries (dual shoulder replacements along with knee and hand surgeries). We discontinued his adalimumab temporarily in 2006 when Michael came down with pneumonia and again in September 2008 when he was hospitalized with a shingles infection that affected his vision.

Michael's health history also included removal of a testicular mass, hernia repair, bilateral knee arthroscopies, and right Achilles tendon repair. After a 2008 shoulder surgery, Michael developed 3 blood clots in his legs and was put on warfarin for a year.

Like many of our patients with RA, Michael had a medical history that included hypertension. However, his condition was well controlled on bisoprolol fumarate and hydrochlorothiazide, and he had no history of hyperlipidemia or family history of early cardiac death. Consequently, the risk of a cardiovascular (CV) event was not high on our list of concerns.

When we saw Michael in early 2009, his disease was still somewhat of a roller coaster, with frequent flares continuing to trouble him. He was just getting back on his RA medications after his most recent shoulder repair. The frustration of years of ups and downs were evident – while Michael rarely complained, the wear and tear of a decade of surgeries and medication modifications were evident just by looking at him.

A few months after this visit, I received a phone call from Michael's brother-in-law. In January 2010, at the age of 54, Michael died in the hospital from complications related to a heart attack.

The story was relayed to me as follows:

Michael was in the hospital visiting his wife, who was having knee replacement surgery. While in the waiting area, Michael suddenly developed a severe headache and intense mid-back pain. As it became more intense, he decided to drive home to take pain medication and lie down. An hour or so later, Michael appeared confused and disoriented, which prompted his mother to call an ambulance. He was admitted to the hospital with an initial diagnosis of encephalopathy due to his immunomodulating medications, headache, and general confusion.

After a normal spinal tap and a resolution of his primary symptoms, Michael was transferred to the telemetry floor, where

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they found a third-degree atrioventricular block. An EKG showed an acute myocardial infarction. Michael soon began experiencing acute shortness of breath, which prompted an echocardiogram. Results showed a large thrombus in his left ventricle.

After years of joint pain, surgeries and fatigue related to his RA, Michael kissed his wife, took off his oxygen, and said that he was tired of fighting.

Michael became increasingly hypoxic. His treatment team formulated a plan of action that included use of a ventilator. But at that point, Michael decided that he had had enough.

After years of joint pain, surgeries and fatigue related to his RA, Michael kissed his wife, took off his oxygen, and said that he was tired of fighting. He died nearly immediately.

As nurses, we often learn important lessons from our patients. Michael taught me the importance of monitoring regularly for CV disease in our patients. While Michael had a few risk factors for CV, including a history of hypertension and poorly controlled seronegative RA, the possibility of a significant CV was rarely foremost in my mind during our conversations.

Looking back, there are so many "what ifs" with Michael.

- What if his seronegative disease had been caught earlier?
- What if his disease had been better controlled?
- What if he took less prednisone less frequently or had found a medication regimen that controlled his disease more adequately so that he could work comfortably without needing daily prednisone?
- What if he had been on prophylaxis aspirin?
- What role did his ophthalmic shingles play in his risk for MI?
- What if the adalimumab had not been held so many times for surgery and infection?

Rheumatology, as with all of medicine, is ever changing. We are lucky to encounter special patients along our professional journey who need us and teach us. We must never forget the Michaels that we meet along the way, as they remind us that RA can be a life-threatening condition that needs to always be treated as the serious disease that it is.

It Takes a Village

by Elizabeth Kirchner, CNP

In nursing school, we are taught to use care plans to help manage our patients' health problems. First, we assess the patient; next, we develop a plan of care; and finally, we implement the plan of care. At the end of this process, the patient response hopefully matches what we were expecting. If not, we go back to step one and start over.

While there is nothing wrong with this systematic approach, taking care of patients in real life rarely adheres to nice, orderly care plans. In fact, it sometimes feels like the more we plan, the more complicated things become.

Such is the case with J.S.

J.S. is a 78-year-old man with a very complicated medical history. He has diverticulitis, benign prostatic hyperplasia, and diabetes mellitus. Add to that unstable angina, hypertension, hyperlipidemia, peripheral artery disease, and coronary artery disease. And a myocardial infarction suffered almost 20 years ago resulting in a coronary artery bypass graft. (Wait, there's more!)

J.S. is a smoker. Finally, he has seropositive rheumatoid arthritis (RA).

J.S. has been coming to our clinic for about 10 years. His RA has proven to be refractory to most of the treatments we have tried. This is no doubt at least partly due to the fact that he refuses to quit smoking despite multiple referrals to smoking cessation clinics and offers of pharmacologic and nonpharmacologic interventions. Since we can't change his behavior, we have to work with what we have. J.S. is currently on methotrexate and folic acid and had, until recently, also been receiving intravenous infliximab every 8 weeks.

At J.S.' most recent visit, we went through all the usual questions as part of our review of symptoms (ie, any signs of infection, recent changes in health, medication updates). J.S. vaguely answered that he had nothing new to report, although it was clear that he was avoiding directly answering our inquiries. When we finally got to the question about recent hospitalizations, the magic lightbulb went on, and J.S. informed us that he had just been discharged from a local hospital after a new case of congestive heart failure (CHF).

At this point, I may have literally banged my head against the wall. Infliximab, as we know, is contraindicated at high doses in patients with moderate-to-severe CHF.[1] While J.S. was unclear about his stage of CHF, we certainly couldn't infuse infliximab without knowing.

So much for our nice, tidy, completely worthless plan of care. And we still had a patient who needed treatment for his RA.

So what did we do? The only thing we could do, really. We sent J.S. home without his infliximab. But we also made sure he had a follow-up appointment scheduled with his cardiologist, started the paperwork to get his outside records faxed to us, and counseled him (again) about the need to quit smoking.

On paper, J.S. looks like a train wreck. But in person, he is really a delightful (if somewhat ornery) gentleman who is managing very well on his own despite issues that would overwhelm the majority of my patients. When I meet someone like J.S., I am reminded that RA is almost never just RA. It brings with it risk factors for so many other conditions, and it is only with constant vigilance and care that we can hope for good outcomes for our patients.

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Hoping for a Home Run, but Ending with a Strikeout

by Jacqueline Fritz, RN, MSN, CNS, RNBC

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Jacqueline Fritz, RN, MSN, CNS, RNBC, is Owner and Coordinator of Education at the Medical Advancement Center in Cypress, CA. Her primary responsibility is working as an advanced practice nurse for a large rheumatology practice where she is involved in patient visits, research programs, and infusion center coordination. In addition, she enjoys speaking, teaching, and learning about immunology.

J.T. was a former professional baseball player who came to our practice after being diagnosed at age 45 with severe rheumatoid arthritis (RA). In the prime of his youth, J.T. played for a number of minor league teams, and even after his baseball career, he remained an avid exercise enthusiast and was a regular at his neighborhood gyms.

Unfortunately, as he neared his 40th birthday, J.T.'s body began to break down, and by the time he came into our office for an evaluation, he was deemed totally disabled.

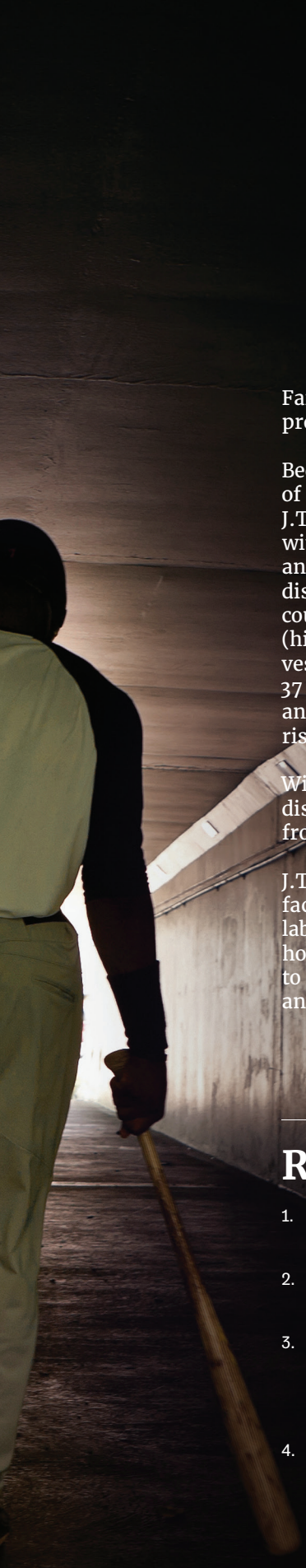
A full workup showed 12 tender and 10 swollen joints, multiple metacarpophalangeal and proximal interphalangeal joint erosions, as well as elevated C-reactive protein (CRP) and erythrocyte sedimentation rates (ESR). In addition, J.T.'s anti-cyclic citrullinated peptide (anti-CCP) and rheumatoid factor (RF) levels both tested positive, as did his anti-Ro/SSA antigens, anti-La/SSB antigens, and anticardiolipin antibodies.

Based on these results, we diagnosed J.T. with Sjögren's syndrome secondary to RA. While he had no previous personal history of cardiovascular disease (CVD), J.T.'s brother had been diagnosed with coronary artery disease in his early 40s and had triple bypass surgery at age 45. Increasing our concerns for development of CVD, J.T. also tested low for testosterone, which can raise the risk of developing CVD by 25%.[1]

When he came to our office, J.T. had been taking prednisone for approximately 10 years to manage his pain. Because of his elevated risk of developing CVD, we hoped to be able to carefully wean J.T. off of steroids, which we never were able to reduce below 10 mg a day.

Unfortunately, J.T. had problems with nearly every medication we threw at him, rapidly going from one to the next every few months. He had gastrointestinal issues with doses of methotrexate greater than 10 mg, and eventually couldn't even tolerate a low dose without having breathing difficulties. He failed to respond to pantoprazole, dexlansoprazole, esomeprazole, and omeprazole. Hydroxychloroquine caused visual changes. Sulfasalazine, even at a low dose of 500 mg BID, caused GI issues.

We then brought out the big guns by moving to the biologics, although we had no greater success. Six months after starting etanercept, J.T. developed pneumonia. Infliximab was effective for only 1 year. He had an allergic reaction to rituximab in the infusion center. Tocilizumab caused too many GI-related side effects. Abatacept was not covered by his insurance.



Failure after failure after failure. It shouldn't be surprising that J.T. became profoundly depressed as a result of all of these disappointments.

Because of his family history of CVD, positive anti-CCP and RF levels, duration of RA, and multiple extra-articular manifestations, we kept a close eye on J.T.'s CVD-related risks during his years being treated within our center. As with management of his RA, we had limited success managing his hemoglobin and hematocrit levels (he was eventually diagnosed with anemia of chronic disease). J.T.'s ESR and CRP rates also remained elevated throughout the course of his care, which is particularly dangerous in patients at risk for CVD (high ESR and CRP levels can promote endothelial dysfunction and structural vessel abnormalities).[2] We were also concerned about J.T.'s very low HDL of 37 md/dL (a risk factor for the development of a coronary artery disease)[3] and his depression, which can increase levels of coronary calcium (another risk factor of CVD).[4]

With J.T., there was challenge after challenge, and disappointment after disappointment. The final blow came when, at age 52, he was found dead from apparent sudden cardiac arrest.

J.T. was a difficult case for a variety of reasons. With so many mitigating factors and variables to control—long-term uncontrolled RA, unfavorable laboratory results, crushing depression, and more—the outcome was not the home run we were seeking. Despite our struggles in J.T.'s care, we are lucky to have fewer strikeouts today with the growing body of information about RA and CVD and the array of treatment options we can provide to our patients.

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Self-Efficacy Assessments: Valuable Tools for Patient Success

by Sheree C. Carter, PhD, RN

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Because patient outcomes are increasingly being used as outcomes measures to determine the success or failure of certain treatment regimens, health behavior modifications, and compliance issues, rheumatology nurses are becoming increasingly more important in developing constructive relationships with patients with chronic diseases such as rheumatoid arthritis (RA).

This issue of *Rheumatology Nurse Practice* concentrates on the intersection between RA and cardiovascular disease, providing you with evidence-based information to serve as a basic educational resource. The concept of self-efficacy fits quite nicely with this topic.

To best serve your patients, you should understand the core attributes of self-efficacy, their clinical significance, and the positive and negative consequences associated with the concept.[1]

Self-efficacy, later coined "social cognitive theory," was first identified as a major concept in social learning theory by psychologist Albert Bandura."[2] The concept behind this theory is that the more positive or higher self-efficacy coping behaviors or beliefs a patient demonstrates, the more likely they are to apply and adhere to certain regimens to achieve specific goals. In other words, if they believe what they do can make a difference, then it will make a difference.

Theoretically, it makes sense. But how can we as rheumatology nurses assess or measure self-efficacy in our patients and, most importantly, move individual patients from negative to positive self-efficacy?

In 2003, a study by Mueller et al found that a fibromyalgia patient's initially low rated self-efficacy can be positively affected when that patient joins a group based on a physiotherapeutic regimen and a psychological focus.[3] This can perhaps in part be attributed to the Hawthorne effect, when those being watched know they are being watched and therefore show the desired effect. We do know that the process of joining a group of others with similar or the same chronic issues sometimes becomes validating to patients and can have an antidepressant effect all to itself. In this scenario, self-efficacy can be thought of as both a process and outcome. In any case, it shows that some learned behavior can result in a positive skew toward self-efficacy.[3,4]

A 2011 study by Knittle et al examined the effects of physical activity goals and self-efficacy beliefs on RA-related pain and quality of life. Data from several measurement scales were collected on 106 participants in the Netherlands. Not surprisingly, patients demonstrating higher self-efficacy at baseline were more likely to achieve physical fitness goals.[4] The takeaway from the study is simple—higher levels of self-efficacy predict higher levels of self-selected goal achievement. Moreover, promoting self-efficacy and helping patients with RA select small, attainable goals can have a positive effect on overall outcomes.

Nurses can have a significant impact on a patient's self-efficacy by spending more focused time helping set short-term, realistic, achievable, and mutually-determined goals. Setting a detailed action plan, creating provisions for accountability and feedback, and having a contingency plan to cope with any barriers that may arise to prevent success is a smart and highly beneficial use of time with patients, especially those with low levels of self-efficacy.

Rheumatology nurses have the ability to serve as the authority on health issues to our patients by providing education, highlighting risk factors for comorbid conditions, encouraging lifestyle and behavioral changes, and referring to self-help groups.

My challenge to you today is to actively evaluate your patients' level of self-efficacy. There are formal instruments such as the

arthritis self-efficacy scale (ASES), the generalized self-efficacy scale (GSES), the Swedish exercise self-efficacy scale (ESES-S), and the rheumatoid arthritis self-efficacy scale (RASE) that may help.[3-8] There are also more focused instruments such as the joint protection self-efficacy scale (JP-SES) or the Marcus and Resnick self-efficacy exercise behavior scale (SEEB).[9,10] ASES and RASE have been more prominently used in clinical trials. Eight of the questions used in ASES are included in Table 1.[3]

Please note that any formal data collection for potential use in publication or a research trial must follow proper institutional review board requirements and adhere to good clinical practice guidelines and regulations. You ***must*** obtain informed consent from patients if you see an opportunity for use of this scale in a future publication. For individual use of the scale for patient-provider communication, however, consent is not required.

Each question on the ASES scale is scored on a simple 1-to-10 range, just like a global assessment rating. These eight questions can be used with patients to assess self-efficacy at a particular point in time. Ask these questions of patients not only for the visit at hand, but also find out how might they have rated themselves in the last week or last month. You may receive valuable assessment information and gain insight on the individualized approach needed. In the end, these tools may assist you in maximizing time with your patients and increase their chances of a successful and positive outcome.

ARTHRITIS SELF-EFFICACY SCALE

(All questions graded on a 1-to-10 scale, with 1=very uncertain and 10=very certain)

1. How certain are you that you can decrease your pain quite a bit?
2. How certain are you that you can keep arthritis pain from interfering with your sleep?
3. How confident are you that you can keep the physical discomfort of your arthritis pain from interfering with the things that you want to do?
4. How certain are you that you can regulate your activity so as to be active without aggravating your arthritis?
5. How confident are you that you can keep the fatigue caused by your arthritis from interfering with the things that you want to do?
6. How certain are you that you can do something to help yourself feel better if you are feeling blue?
7. As compared to other people with arthritis like yours, how certain are you that you can manage arthritis pain during your daily activities?
8. How certain are you that you can deal with the frustration of arthritis?

Table 1 *Adapted from Appendix A in Mueller A, Hartmann M, Mueller K, Eich W. Validation of the arthritis self-efficacy short-form scale in German fibromyalgia patients. Eur J Pain. 2003;7(2):163-171.*

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